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=> s RECEPTOR (P) (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND) (P) (SCREEN? OR
BIND? OR ASSAY) and orphan
   3 FILES SEARCHED...
L1        2546 RECEPTOR (P) (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND) (P)
               (SCREEN? OR BIND? OR ASSAY) AND ORPHAN

=> s l1 and py<=1998
   2 FILES SEARCHED...
L2         912 L1 AND PY<=1998

=> dup rem l2
PROCESSING COMPLETED FOR L2
L3          284 DUP REM L2 (628 DUPLICATES REMOVED)

=> s l3 and (gpcr or g adj protein?coupled adj receptor)
'?' TRUNCATION SYMBOL NOT VALID WITHIN 'PROTEIN?COUPLED'
The truncation symbol ?  may be used only at the end of a search
term.  To specify a variable character within a word use '!', e.g.,
'wom!n' to search for both 'woman' and 'women'.  Enter "HELP
TRUNCATION" at an arrow prompt (=>) for more information.

=> s l3 and (gpcr or g (w) protein)
L4          33 L3 AND (GPCR OR G (W) PROTEIN)

=> d 1-33 bib ab

L4   ANSWER 1 OF 33     MEDLINE on STN
AN   2004192058      IN-PROCESS Full-text
DN   PubMed ID: 12671309
TI   The Potential Role of the Transcription Factor RZR/ROR as a Mediator of
     Nuclear Melatonin Signaling.
AU   Wiesenberg I; Missbach M; Carlberg C
CS   Pharma Research, NOVARTIS, Basel, Switzerland.
SO   Restorative neurology and neuroscience, (1998) 12 (2-3) 143-50.
     Journal code: 9005499. ISSN: 0922-6028.
CY   Netherlands
DT   Journal; Article; (JOURNAL ARTICLE)
LA   English
FS   IN-DATA-REVIEW; IN-PROCESS; NONINDEXED
ED   Entered STN: 20040417
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Last Updated on STN: 20041219

AB The pineal gland hormone melatonin is well known as a regulator of circadian rhythmicity, but has also other functions in the central nervous system as well as in the periphery including the maturation of neurons and the regulation of cellular growth and differentiation. Three mechanisms of the hormone's action are currently discussed: a membrane signaling pathway, a nuclear signaling pathway and a **receptor**-independent radical scavenging function. Melatonin membrane **receptors** are seven transmembrane **receptors** and mediate their functions through a G-**protein**-coupled second messenger pathway. Nuclear melatonin signaling seems to be mediated via the transcription factor RZR/ROR, which is an **orphan** member of the nuclear **receptor** superfamily. The widespread distribution of the a-subtype of RZR/ROR suggests that this **receptor** may be an important mediator of those effects of melatonin that can not be explained by membrane **receptors** or radical scavenging. Interestingly, natural RZR/RORa "knock-out" mice (staggerer) show severe defects in the development of cerebellar Purkinje cells, a reduced body weight and immunological defects. RZR/ROR **binds** as a monomer to DNA, but also forms homodimers on appropriate **binding** sites. Natural RZR/ROR **binding** sites have been identified in the regulatory regions of many genes. 5-lipoxygenase, p21WAF1/CIP1, apolipoprotein A-1, N-myc and Purkinje cell protein 2 may be functionally important target genes. On some of these **binding** sites RZR/ROR competes with other members of the nuclear **receptor** superfamily (e.g., COUP-TF, RAR and Rev-ErbA) indicating a cross-talk between these transcription factors RZR/ROR often shows in transient transfection **assays** a high constitutive, i.e. **ligand**-independent activity. However, under conditions of low constitutive activity a significant and specific stimulation of RZR/ROR by low nanomolar concentrations of melatonin and structurally novel class of thiazolidinediones (lead structure: CGP52608) has been observed. Taken together, the effects of melatonin on transcriptional regulation clearly depend on the expression fo RZR/ROR and support the concept that the **receptor** is a mediator of nuclear melatonin signaling.

L4 ANSWER 2 OF 33 MEDLINE on STN
AN 1999098533 MEDLINE Full-text
DN PubMed ID: 9884064
TI **Orphan G-protein**-coupled receptors: the next generation of drug targets?.
AU Wilson S; Bergsma D J; Chambers J K; Muir A I; Fantom K G; Ellis C; Murdock P R; Herrity N C; Stadel J M
CS SmithKline Beecham Pharmaceuticals, Harlow, Essex.
SO British journal of pharmacology, (**1998 Dec**) 125 (7) 1387-92.
Ref: 25
Journal code: 7502536. ISSN: 0007-1188.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199904
ED Entered STN: 19990413
Last Updated on STN: 20000303
Entered Medline: 19990401

AB The pharmaceutical industry has readily embraced genomics to provide it with new targets for drug discovery. Large scale DNA sequencing has allowed the identification of a plethora of DNA sequences distantly related to known G **protein**-coupled **receptors** (**GPCRs**), a superfamily of **receptors** that have a proven history of being excellent therapeutic targets. In most cases the extent of sequence homology is insufficient to assign these '**orphan**' **receptors**

to a particular **receptor** subfamily. Consequently, reverse molecular pharmacological and functional genomic strategies are being employed to identify the activating **ligands** of the cloned **receptors**. Briefly, the reverse molecular pharmacological methodology includes cloning and expression of **orphan GPCRs** in mammalian cells and **screening** these cells for a functional response to cognate or surrogate **agonists** present in biological extract preparations, peptide libraries, and complex **compound** collections. The functional genomics approach involves the use of 'humanized yeast cells, where the yeast **GPCR** transduction system is engineered to permit functional expression and coupling of human **GPCRs** to the endogenous signalling machinery. Both systems provide an excellent platform for identifying novel **receptor ligands**. Once activating **ligands** are identified they can be used as pharmacological tools to explore **receptor** function and relationship to disease.

L4 ANSWER 3 OF 33 MEDLINE on STN
AN 1999084057 MEDLINE Full-text
DN PubMed ID: 9866825
TI Effects of newly isolated opioid peptides on **G-protein** activation: usefulness of [35S] GTP gamma S binding study and its practical application.
AU Narita M
CS Department of Anesthesiology, Medical College of Wisconsin, Milwaukee 53226, USA.
SO Nihon shinkei seishin yakurigaku zasshi = Japanese journal of psychopharmacology, **(1998 Aug)** 18 (4) 107-16. Ref: 58
Journal code: 9509023. ISSN: 1340-2544.
CY Japan
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA Japanese
FS Priority Journals
EM 199902
ED Entered STN: 19990316
Last Updated on STN: 20000303
Entered Medline: 19990226
AB Recent cloning and expression studies have revealed that the opioid mu-, delta-, kappa- and **orphan receptors** are seven-transmembrane domain **receptors** whose actions are mediated through activation of guanine nucleotide **binding** protein (**G-protein**). The activation of **G-proteins** by the opioid **receptor** can be measured by assessing **agonist** stimulation of membrane **binding** of the non-hydrolyzable analog of guanosine triphosphate (GTP), guanosine-5'-O-(3-[35S] thio) triphosphate ([35S] GTP gamma S). Our recent data suggest that 1) the level of spinal mu-, delta-, kappa- and **orphan-receptor agonist**-stimulated [35S] GTP gamma S **binding** closely parallels that of **receptor binding** densities, 2) the neuroanatomical distribution of opioid **agonist**-stimulated [35S] GTP gamma S **binding** relates to **receptor binding** distribution, 3) newly isolated opioid peptides, endomorphin-1 and -2, can activate **G-proteins** by specific stimulation of mu-**receptors** and act as partial **agonists** with moderate catalytic efficacies, 4) mu- **receptor** densities could be rate-limiting steps in the **G -protein** activation by mu-**agonists** in the spinal cord region. In conclusion, opioid **agonist**-stimulated [35S] GTP gamma S **binding** can provide a functional method to localize **receptors** not only by their ability to **bind ligands**, but also according to their ability to activate an intracellular signal transducer.

L4 ANSWER 4 OF 33 MEDLINE on STN

AN 1998341734 MEDLINE Full-text
DN PubMed ID: 9678594
TI Functional genomics: the search for novel neurotransmitters and neuropeptides.
AU Civelli O
CS Department of Pharmacology, University of California, Irvine 92697-4625, USA.. ocivelli@uci.edu
SO FEBS letters, (1998 Jun 23) 430 (1-2) 55-8. Ref: 40
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199808
ED Entered STN: 19980903
Last Updated on STN: 19980903
Entered Medline: 19980825
AB Functional genomics can be defined as the search for the physiological role of a gene for which only its primary sequence is known. Most of the genes encoding proteins containing seven hydrophobic stretches code for G **protein**-coupled **receptors** (**GPCRs**). Although many of these have been shown to interact with known natural **ligands**, several **bind ligands** which have not been thus far isolated. These are the so-called **orphan GPCRs**. As an example of functional genomics, an '**orphan receptor** strategy' has been developed to identify the natural **ligands** of **orphan GPCRs**. The application of this strategy is bound to revolutionize our understanding of the diversity of the primary messengers which modulate synaptic transmission. This review discusses the basic concepts and some of the particular problems associated with the **orphan receptor** strategy. The strategy's potential is exemplified by its successes which culminated in the discovery of the neuropeptides 'orphanin FQ/nociceptin' and 'orexins/hypocretins'. The steps that led to the characterization of these neuropeptides are discussed as are some of the further studies that have addressed the roles of these neuropeptides. To conclude, some of the implications of the application of the **orphan receptor** strategy are discussed.

L4 ANSWER 5 OF 33 MEDLINE on STN
AN 1998264639 MEDLINE Full-text
DN PubMed ID: 9603476
TI ChemR23, a putative chemoattractant receptor, is expressed in monocyte-derived dendritic cells and macrophages and is a coreceptor for SIV and some primary HIV-1 strains.
AU Samson M; Edinger A L; Stordeur P; Rucker J; Verhasselt V; Sharron M; Govaerts C; Mollereau C; Vassart G; Doms R W; Parmentier M
CS IRIBHN, Universite libre de Bruxelles, Belgium.
NC AI-35383 (NIAID)
AI-38225 (NIAID)
AI-40880 (NIAID)
SO European journal of immunology, (1998 May) 28 (5) 1689-700.
Journal code: 1273201. ISSN: 0014-2980.
CY GERMANY: Germany, Federal Republic of
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; AIDS
OS GENBANK-Y14838
EM 199806
ED Entered STN: 19980611

Last Updated on STN: 19980611
Entered Medline: 19980603

AB Leukocyte chemoattractants act through a rapidly growing subfamily of G **protein**-coupled **receptors**. We report the cloning of a novel human gene encoding an **orphan receptor** (ChemR23) related to the C3a, C5a and formyl Met-Leu-Phe **receptors**, and more distantly to the subfamilies of chemokine **receptors**. ChemR23 transcripts were found to be abundant in monocyte-derived dendritic cells and macrophages, treated or not with LPS. Low expression could also be detected by reverse transcription-PCR in CD4+ T lymphocytes. The gene encoding ChemR23 was assigned by radiation hybrid mapping to the q21.2-21.3 region of human chromosome 12, outside the gene clusters identified so far for chemoattractant **receptors**. Given the increasing number of chemoattractant **receptors** used by HIV-1, HIV-2 and SIV as coreceptors, ChemR23 was tested in fusion **assays** for potential coreceptor activity by a range of viral strains. None of the tested HIV-2 strains made use of ChemR23 as a coreceptor, but several SIV strains (SIVmac316, SIVmac239, SIVmacl7E-Fr and SIVsm62A), as well as a primary HIV-1 strain (92UG024-2) used it efficiently. ChemR23 therefore appears as a coreceptor for immunodeficiency viruses that does not belong to the chemokine **receptor** family. It is also a putative chemoattractant **receptor** relatively specific for antigen-presenting cells, and it could play an important role in the recruitment or trafficking of these cell populations. Future work will be required to identify the **ligand**(s) of this new G **protein**-coupled **receptor** and to define its precise role in the physiology of dendritic cells and macrophages.

L4 ANSWER 6 OF 33 MEDLINE on STN
AN 1998244535 MEDLINE Full-text
DN PubMed ID: 9585136
TI Molecular mechanisms of G **protein**-coupled receptor desensitization and resensitization.
AU Ferguson S S; Zhang J; Barak L S; Caron M G
CS John P. Robarts Research Institute and Department of Physiology, University of Western Ontario, London.
SO Life sciences, **(1998)** 62 (17-18) 1561-5. Ref: 22
Journal code: 0375521. ISSN: 0024-3205.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199805
ED Entered STN: 19980609
Last Updated on STN: 20000303
Entered Medline: 19980526

AB Beta-arrestin proteins play a dual role in regulating G **protein**-coupled **receptor** (GPCR) responsiveness by contributing to both **receptor** desensitization and internalization. Recently, beta-arrestins were also shown to be critical determinants for beta2-adrenergic **receptor** (beta2AR) resensitization. This was demonstrated by overexpressing wild-type beta-arrestins to rescue the resensitization-defect of a beta2AR (Y326A) mutant (gain of function) and overexpressing a dominant-negative beta-arrestin inhibitor of beta2AR sequestration to impair beta2AR dephosphorylation and resensitization (loss of function). Moreover, the ability of the beta2AR to resensitize in different cell types was shown to be dependent upon beta-arrestin expression levels. To further study the mechanisms underlying beta-arrestin function, green fluorescent protein was coupled to beta-arrestin2 (beta arr2GFP), thus allowing the real-time visualization of the **agonist**-dependent trafficking of beta-arrestin in living cells. Beta arr2GFP

translocation from the cytoplasm to the plasma membrane proceeded with a time course, sensitivity and specificity that was indistinguishable from the most sensitive second messenger readout systems. Beta arr2GFP translocation was GRK-dependent and was demonstrated for 16 different **ligand**-activated **GPCRs**. Because beta-arrestin **binding** is a common divergent step in **GPCR** signalling, this **assay** represents a universal methodology for **screening orphan receptors**, GRK inhibitors and novel **GPCR ligands** . Moreover, beta arr2GFP provides a valuable new tool to dissect the biological function and regulation of beta-arrestin proteins.

L4 ANSWER 7 OF 33 MEDLINE on STN
AN 1998115534 MEDLINE Full-text
DN PubMed ID: 9455999
TI Presence of nociceptin (orphanin FQ) receptors in rat retina: comparison with receptors in striatum.
AU Makman M H; Dvorkin B
CS Department of Biochemistry, Albert Einstein College of Medicine, Bronx, NY 10461, USA.
SO European journal of pharmacology, (**1997 Nov** 5) 338 (2) 171-6.
Journal code: 1254354. ISSN: 0014-2999.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199803
ED Entered STN: 19980319
Last Updated on STN: 19980319
Entered Medline: 19980312
AB Nociceptin (orphanin FQ), a heptadecapeptide with some sequence homology to dynorphin A, has been proposed as an endogenous **ligand** for a previously cloned **orphan receptor** with significant homology to opioid **receptors**. Utilizing [(125)I][Tyr14]nociceptin as **ligand**, saturable and high affinity nociceptin **binding** sites were detected and characterized in rat retina and striatum. For retina, Bmax = 44.0 +/- 4.5 fmol/mg and Kd = 32.4 +/- 2.7 pM; for striatum, Bmax = 51.6 +/- 7.7 fmol/mg and Kd = 98.6 +/- 11.3 pM. In competition studies, nociceptin bound with picomolar affinity, dynorphin A with nanomolar affinity, naloxone and dynorphan A-(1-8) with micromolar affinity, while [des-Tyr1]dynorphin (dynorphin A-(2-17)), several other opioids, morphine and benzomorphans failed to compete for **binding** at 1-10 microM. Gpp(NH)p plus NaCl markedly decreased **binding**, consistent with involvement of a **G protein**-linked **receptor**. It is concluded that rat retina contains nociceptin **receptors** similar in concentration to those present in striatum. Properties of both the retinal and the striatal **receptors** are similar to those previously found for rat hypothalamus.

L4 ANSWER 8 OF 33 MEDLINE on STN
AN 1998021380 MEDLINE Full-text
DN PubMed ID: 9378852
TI Anatomical distribution of mu, delta, and kappa opioid- and nociceptin/orphanin FQ-stimulated [35S]guanylyl-5'-O-(gamma-thio)-triphosphate binding in guinea pig brain.
AU Sim L J; Childers S R
CS Department of Physiology and Pharmacology, Center for the Neurobiological Investigation of Drug Abuse, Bowman Gray School of Medicine, Wake Forest University, Winston-Salem, North Carolina 27157, USA.
NC CA-12197 (NCI)
DA-00287 (NIDA)
DA-02904 (NIDA)

SO Journal of comparative neurology, (1997 Oct 6) 386 (4) 562-72.
Journal code: 0406041. ISSN: 0021-9967.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 20000303
Entered Medline: 19971110
AB An in vitro autoradiographic technique has recently been developed to visualize **receptor**-activated **G-proteins** by using **agonist**-stimulated [35S]guanylyl-5'-O-(gamma-thio)- triphosphate ([35S]GTPgammaS) **binding** in the presence of excess guanosine 5'-diphosphate. This technique was used to localize opioid-activated **G-proteins** in guinea pig brain, a species that contains the three major types of opioid **receptors.** This study used selective mu, delta, and kappa opioid **agonists** as well as nociceptin or orphanin FQ (N/OFQ) peptide, an endogenous **ligand** for an **orphan** opioid **receptor**-like (ORL1) **receptor**, to stimulate [35S]GTPgammaS **binding** in guinea pig brain sections. Opioid **receptor** specificity was confirmed by blocking **agonist**-stimulated [35S] GTPgammaS **binding** with the appropriate **antagonists.** In general, the distribution of **agonist**-stimulated [35S]GTPgammaS **binding** correlated with previous reports of **receptor binding** autoradiography, although quantitative differences suggest regional variations in **receptor** coupling efficiency. Mu, delta, and kappa opioid-stimulated [35S]GTPgammaS **binding** was found in the caudate-putamen, nucleus accumbens, amygdala, and hypothalamus. Mu-stimulated [35S]GTPgammaS **binding** predominated in the hypothalamus, amygdala, and brainstem, whereas kappa-stimulated [35S]GTPgammaS **binding** was particularly high in the substantia nigra and cortex and was moderate in the cerebellum. N/OFQ-stimulated [35S] GTPgammaS **binding** was highest in the cortex, hippocampus, and hypothalamus and exhibited a unique anatomical distribution compared with opioid-stimulated [35S]GTPgammaS **binding.** The present study extends previous reports on opioid and ORL1 **receptor** localization by anatomically demonstrating functional activity produced by mu, delta, and kappa opioid and ORL1 **receptor** activation of **G-proteins.**

L4 ANSWER 9 OF 33 MEDLINE on STN
AN 1998010570 MEDLINE Full-text
DN PubMed ID: 9346876
TI A beta-arrestin/green fluorescent protein biosensor for detecting **G protein**-coupled receptor activation.
AU Barak L S; Ferguson S S; Zhang J; Caron M G
CS Howard Hughes Medical Institute Laboratories and Department of Cell Biology, Duke University Medical Center, Durham, North Carolina 27710, USA.
NC HL 03422 (NHLBI)
NS 19576 (NINDS)
SO Journal of biological chemistry, (1997 Oct 31) 272 (44) 27497-500.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199712
ED Entered STN: 19980109
Last Updated on STN: 20000303
Entered Medline: 19971209
AB **G protein**-coupled **receptors** (GPCR)

represent the single most important drug targets for medical therapy, and information from genome sequencing and genomic data bases has substantially accelerated their discovery. The lack of a systematic approach either to identify the function of a new GPCR or to associate it with a cognate ligand has added to the growing number of orphan receptors. In this work we provide a novel approach to this problem using a beta-arrestin2/green fluorescent protein conjugate (betaarr2-GFP). It provides a real-time and single cell based assay to monitor GPCR activation and GPCR-G protein-coupled receptor kinase or GPCR-arrestin interactions. Confocal microscopy demonstrates the translocation of betaarr2-GFP to more than 15 different ligand-activated GPCRs. These data clearly support the common hypothesis that the beta-arrestin binding of an activated receptor is a convergent step of GPCR signaling, increase by 5-fold the number of GPCRs known to interact with beta-arrestins, demonstrate that the cytosol is the predominant reservoir of biologically active beta-arrestins, and provide the first direct demonstration of the critical importance of G protein -coupled receptor kinase phosphorylation to the biological regulation of beta-arrestin activity and GPCR signal transduction in living cells. The use of betaarr2-GFP as a biosensor to recognize the activation of pharmacologically distinct GPCRs should accelerate the identification of orphan receptors and permit the optical study of their signal transduction biology intractable to ordinary biochemical methods.

L4 ANSWER 10 OF 33 MEDLINE on STN
AN 97467951 MEDLINE Full-text
DN PubMed ID: 9324949
TI A bioluminescent assay for agonist activity at potentially any G-protein-coupled receptor.
AU Stables J; Green A; Marshall F; Fraser N; Knight E; Sautel M; Milligan G; Lee M; Rees S
CS Receptor Systems Unit, Glaxo Wellcome Research and Development, Stevenage, Herts, United Kingdom.
SO Analytical biochemistry, (1997 Oct 1) 252 (1) 115-26.
Journal code: 0370535. ISSN: 0003-2697.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 20021218
Entered Medline: 19971117
AB Transient expression of apoaequorin in Chinese hamster ovary (CHO) cells and reconstitution with the co-factor coelenterazine resulted in a large, concentration-dependent agonist-mediated luminescent response following cotransfection with the endothelin ETA, angiotensin ATII, thyrotropin-releasing hormone (TRH), and neurokinin NK1 receptors , all of which interact pre-dominantly with the G alpha q-like phosphoinositidase-linked G-proteins. A substantially greater luminescence was obtained with mitochondrially targeted apoaequorin compared to cytoplasmically expressed apoaequorin. To generate a system amenable for the study of agonist activity at virtually any G-protein-coupled receptor the alpha subunit of the receptor promiscuous G-protein G alpha 16 was either transiently or stably expressed in CHO cells together with apoaequorin. In cells expressing G alpha 16, but not in its absence, agonists at a series of receptors which normally interact with either G alpha s or G alpha i were now able to cause a luminescent response from mitochondrially targeted apoaequorin. In the case of the A1 adenosine receptor, this response was clearly a result of activation of G alpha 16 and not a consequence of the release of the G alpha i-associated beta/gamma

complex, as the luminescent response was unaffected by pertussis toxin treatment of the cells, whereas **agonist**-mediated inhibition of adenylyl cyclase activity was attenuated. These studies describe the use of coexpressed apoaequorin as a reporter for **G-protein**-coupled **receptor** -mediated calcium signaling. Furthermore, coexpression of G alpha 16 and apoaequorin provides a basis for a generic mammalian cell microplate **assay** for the assessment of **agonist** action at virtually any **G-protein**-coupled **receptor**, including **orphan receptors** for which the physiological signal transduction mechanism may be unknown.

L4 ANSWER 11 OF 33 MEDLINE on STN
AN 97438287 MEDLINE Full-text
DN PubMed ID: 9292776
TI Molecular approaches to receptors as targets for drug discovery.
AU Herz J M; Thomsen W J; Yarbrough G G
CS Applied Receptor Sciences, Mill Creek, WA 98012, USA.
SO Journal of receptor and signal transduction research, **(1997 Sep)** 17 (5) 671-776. Ref: 275
Journal code: 9509432. ISSN: 1079-9893.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, ACADEMIC)
LA English
FS Priority Journals
EM 199711
ED Entered STN: 19971224
Last Updated on STN: 20000303
Entered Medline: 19971104
AB The cloning of a great number of **receptors** and channels has revealed that many of these targets for drug discovery can be grouped into superfamilies based on sequence and structural similarities. This review presents an overview of how molecular biological approaches have revealed a plethora of **receptor** subtypes, led to new definitions of subtypes and isoforms, and played a role in the development of high selective drugs. Moreover, the diversity of subtypes has molded current views of the structure and function of **receptor** families. Practical difficulties and limitations inherent in the characterization of the **ligand binding** and signaling properties of expressed recombinant **receptors** are discussed. The importance of evaluating drug-**receptor** interactions that differ with temporally transient and distinct **receptor** conformational states is emphasized. Structural motifs and signal transduction features are presented for the following major **receptor** superfamilies: **ligand**-gated ion channel, voltage-dependent ion channel, G -**protein** coupled, **receptor** tyrosine-kinase, **receptor** protein tyrosine-phosphatase, cytokine and nuclear hormone. In addition, a prototypic **receptor** is analyzed to illustrate functional properties of a given family. The review concludes with a discussion of future directions in **receptor** research that will impact drug discovery, with a specific focus on **orphan receptors** as targets for drug discovery. Methods for classifying **orphan receptors** based upon homologies with members of existing superfamilies are presented together with molecular approaches to the greater challenge of defining their physiological roles. Besides revealing new **orphan receptors**, the human genome sequencing project will result in the identification of an abundance of novel **receptors** that will be molecular targets for the development of highly selective drugs. These findings will spur the discovery and development of an exciting new generation of **receptor**-subtype specific drugs with enhanced therapeutic specificity.

L4 ANSWER 12 OF 33 MEDLINE on STN

AN 97404870 MEDLINE Full-text
DN PubMed ID: 9261564
TI Molecular characterization and functional expression of opioid receptor-like1 receptor.
AU Wu Y L; Pu L; Ling K; Zhao J; Cheng Z J; Ma L; Pei G
CS Shanghai Institute of Cell Biology, Chinese Academy of Sciences, China.
SO Cell research, (1997 Jun) 7 (1) 69-77.
Journal code: 9425763. ISSN: 1001-0602.
CY China
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199709
ED Entered STN: 19971008
Last Updated on STN: 20000303
Entered Medline: 19970919
AB The opioid **receptor**-like1 **receptor** (ORL1), an **orphan receptor** whose human and murine complementary DNAs, has been characterized recently. ORL1 transcripts are particularly abundant in the central nervous system. We demonstrated that ORL1 expressed in human neuroblastoma SK-N-SH and SH-SY5Y cell lines by radioligand **binding assay**, reverse transcription polymerase chain reaction (RT-PCR) and Northern analysis in the present study. Stimulation with ORL1 specific **agonist**, nociceptin/orphanin FQ, increased [35S]GTP gamma S **binding** to SK-N-SH cell membranes (EC50 = 14 +/- 0.45 nM), and attenuated forskolin-stimulated accumulation of cellular cAMP (EC50 = 0.80 +/- 0.45 nM), indicative that activation of ORL1 activates G **proteins** and inhibits adenylyl cyclase. Activation of ORL1 **receptor** was also accessed using CHO:hORL1 cell line by microphysiometer. Treatment of nociceptin/orphanin FQ increased extracellular acidification rate significantly.

L4 ANSWER 13 OF 33 MEDLINE on STN
AN 97382456 MEDLINE Full-text
DN PubMed ID: 9240460
TI The 6H1 **orphan** receptor, claimed to be the p2y5 receptor, does not mediate nucleotide-promoted second messenger responses.
AU Li Q; Schachter J B; Harden T K; Nicholas R A
CS Department of Pharmacology, University of North Carolina at Chapel Hill, 27599-7365, USA.
NC GM38213 (NIGMS)
SO Biochemical and biophysical research communications, (1997 Jul 18) 236 (2) 455-60.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199708
ED Entered STN: 19970908
Last Updated on STN: 19970908
Entered Medline: 19970828
AB An **orphan** G **protein**-coupled **receptor** , termed 6H1, with approximately 30% sequence identity to P2Y **receptors** has been proposed to be a P2Y **receptor** (p2y5) based solely on a radioligand **binding assay** with [35S]dATP alphaS [Webb et al. (1997) Biochem. Biophys. Res. Commun. 219:105-110]. Previous work in our laboratory has shown that [35S]-dATP alphaS is not a general **ligand** for P2Y **receptors**, and thus inclusion of the p2y5 **receptor** in the family of P2Y **receptors** is questionable. To define unambiguously whether the p2y5 **receptor** is a P2Y **receptor**, we have cloned the turkey homologue of the chick p2y5 **receptor**. Sequence analysis indicated that the turkey **receptor** contains an

additional 32 amino acids at its carboxy terminus compared to the published chick sequence. HA epitope-tagged turkey p2y5 **receptors** were stably expressed in 1321N1 human astrocytoma cells, and cells shown to express the HA-tagged p2y5 **receptor** by an intact cell-based ELISA were used to determine whether changes in second messenger levels occurred in response to a series of nucleotides. ATP, ADP, UTP, UDP, dATP alphaS, and A2P4 had no effect on either inositol phosphate or cyclic AMP concentrations in cells expressing the p2y5 **receptor**. Robust inositol phosphate and cyclic AMP responses occurred to other **G protein-coupled receptors** expressed in 1321N1 cells, which indicate that these cells contain all of the necessary signaling components to generate these second messenger responses. These data indicate that the 6H1/p2y5 **receptor** is not a member of the P2Y **receptor** family of signaling proteins.

L4 ANSWER 14 OF 33 MEDLINE on STN
AN 97366605 MEDLINE Full-text
DN PubMed ID: 9223435
TI Cloning of a human heptahelical receptor closely related to the P2Y5 receptor.
AU Janssens R; Boeynaems J M; Godart M; Communi D
CS Institute of Interdisciplinary Research, School of Medicine, Erasme Hospital, Universite Libre de Bruxelles, Brussels, Belgium.
SO Biochemical and biophysical research communications, (1997 Jul 9) 236 (1) 106-12.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-AF005419
EM 199708
ED Entered STN: 19970813
Last Updated on STN: 19980206
Entered Medline: 19970807
AB The 6H1 **receptor** cloned from activated chicken T cells was initially considered an **orphan** G-coupled **receptor**, but was later included in the P2Y family of **receptors** for purine and pyrimidine nucleotides on the basis of a significant amino acid identity and was renamed P2Y5. Analysis of the expressed sequence tag database revealed the presence of a related sequence exhibiting 63% amino acid identity with this **receptor**. Starting from this partial sequence, we have isolated a complete clone and identified a 1113 base pair open reading frame encoding a new G-coupled **receptor** that we have called P2Y5-like. This sequence exhibits 61% identity with the chicken P2Y5 sequence and 30-33% with other P2Y subtypes. A construct encoding this P2Y5-like **receptor** was transfected into COS-7, 1321N1, and CHO-K1 cells, and expression was documented by Northern blotting. None of the 40 nucleotides and nucleosides tested was able to elicit a response in any of four functional **assays**: inositol phosphate formation, stimulation or inhibition of cAMP formation, and extracellular acidification measured with a microphysiometer. These results suggest either that the natural **ligand** of the P2Y5-like **receptor** is an uncommon nucleotide or alternatively that despite its structural similarity to the P2Y family it is not a nucleotide **receptor**.

L4 ANSWER 15 OF 33 MEDLINE on STN
AN 97312546 MEDLINE Full-text
DN PubMed ID: 9168987
TI Molecular cloning and tissue expression of a novel **orphan** **G protein-coupled** receptor from rat lung.
AU Bonini J A; Anderson S M; Steiner D F

CS Howard Hughes Medical Institute, University of Chicago, Illinois 60637, USA.. jabonini@midway.uchicago.edu
SO Biochemical and biophysical research communications, **(1997 May 8)** 234 (1) 190-3.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U92802
EM 199706
ED Entered STN: 19970709
Last Updated on STN: 20000303
Entered Medline: 19970624
AB **G protein**-coupled **receptors** transduce the signal of a wide variety of hormones, neurotransmitters, cytokines, and other molecules across the cell membrane to elicit the corresponding response inside the target cells. We describe in this paper the molecular cloning and tissue distribution of a novel rat **G protein** -coupled **receptor**, GPR41, with highest homology to the human **orphan G protein**-coupled **receptor** DRY12. A lower degree of homology was seen with the **receptors** for bradykinin, angiotensin, and IL8. The expression of GPR41 appears to be the highest in brain and lung tissues, with lesser expression in heart, skeletal muscle, and kidney, as assayed by northern blotting. No GPR41 message was seen in spleen, liver, or testes. GPR41 failed to **bind** any of the **ligands** tested.

L4 ANSWER 16 OF 33 MEDLINE on STN
AN 97165887 MEDLINE Full-text
DN PubMed ID: 9013780
TI Cloning, characterization, and chromosomal localization of rec1.3, a member of the **G-protein**-coupled receptor family highly expressed in brain.
AU Macrae A D; Premont R T; Jaber M; Peterson A S; Lefkowitz R J
CS Department of Medicine (Cardiology), Howard Hughes Medical Institute, Durham, NC 27710, USA.
NC HL 16037 (NHLBI)
SO Brain research.. Molecular brain research, **(1996 Dec)** 42 (2) 245-54.
Journal code: 8908640. ISSN: 0169-328X.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U18405; GENBANK-U48235; GENBANK-U48236
EM 199704
ED Entered STN: 19970424
Last Updated on STN: 20000303
Entered Medline: 19970416
AB During a project to identify **G-protein**-coupled **receptors** (GPCR) expressed within taste buds, we have isolated a novel **receptor**-like sequence. The full length sequence of this **receptor** (rec1.3) has been obtained in both cow and mouse. Rec1.3 bears little sequence similarity to any **GPCR** whose **ligand** is known: the closest identity (33%) is to the **orphan receptor** edg-1. In cow, rec1.3 is expressed most prominently in the brain, with moderate expression in testis and tongue; in the mouse the expression is more widespread. No specific **binding** for a range of **ligands** was detected when the mouse coding sequence was expressed in eukaryotic cells. In situ hybridization showed that rec1.3 is widely expressed throughout the mouse brain and is highly expressed in localized regions of the hindbrain, midbrain and hypothalamus. The rec1.3

gene was localized to the centromeric region of chromosome 4 in mouse, a region associated with neonatal seizures.

L4 ANSWER 17 OF 33 MEDLINE on STN
AN 96430916 MEDLINE Full-text
DN PubMed ID: 8833998
TI Cloning and characterization of a new member of the G-**protein** coupled receptor EDG family.
AU Masana M I; Brown R C; Pu H; Gurney M E; Dubocovich M L
CS Department of Molecular Pharmacology and Biological Chemistry, Northwestern University Medical School, Chicago, IL 60611, USA.
NC MH42922 (NIMH)
NS07140 (NINDS)
SO Receptors & channels, **(1995)** 3 (4) 255-62.
Journal code: 9315376. ISSN: 1060-6823.
CY Switzerland
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U18405
EM 199611
ED Entered STN: 19961219
Last Updated on STN: 20000303
Entered Medline: 19961127
AB We report here the cloning of a new member of the endothelium differentiation gene (edg) subfamily of **G-protein** -coupled **receptors.** This novel cDNA sequence was cloned from the ovine pars tuberalis using a reverse transcriptase polymerase chain reaction (RT-PCR) amplification with degenerate primers homologous to the highly conserved II and VII transmembrane domains of the **G- protein** coupled **receptor** gene family. The PCR product was random primed with 32P and used as a probe to **screen** a size-selected cDNA ovine pars tuberalis library, which resulted in the isolation of a single clone of 2700 bp. This novel sequence was named edg-2, because its nucleic acid sequence was 55% homologous over 501nt overlap to an **orphan** sequence cloned from human endothelial cells, the endothelial differentiation gene, edg-1. The highest degree of aminoacid homology (42%) occurs in the seven putative transmembrane domains, particularly between the transmembrane domains III and VI (53% and 64%, respectively). The intervening hydrophilic domains are short and there are numerous putative phosphorylation sites for Ser/Thr-protein kinases in the second and third intracellular loop and in the COOH-terminal domain. Through Northern analysis of total RNA, low levels of at least four transcripts of 2.3, 2.5, 3.2 and 4 kb were found in sheep cerebral cortex and a 4.2 kb transcript was observed in NIH/3T3 fibroblasts. In addition, edg-2 transcripts (415 bp) were amplified by RT-PCR from pars tuberalis, cerebral blood vessels, hypothalamus, and retina. Serum stimulation of Chinese hamster ovary (CHO) cells expressing the edg-2 **receptor** resulted in increased cell proliferation, as measured by [3H]-thymidine incorporation. Edg-1 and edg-2 appear to be distinct genes that may encode protein products that **bind** the same or related **ligand.**

L4 ANSWER 18 OF 33 MEDLINE on STN
AN 96228068 MEDLINE Full-text
DN PubMed ID: 8647286
TI Cloning of a melatonin-related receptor from human pituitary.
AU Reppert S M; Weaver D R; Ebisawa T; Mahle C D; Kolakowski L F Jr
CS Laboratory of Developmental Chronobiology, Massachusetts General Hospital, Boston, MA 02114,. USA.reppert@helix.mgh.harvard.edu
NC R37 HD14427 (NICHD)

SO FEBS letters, (1996 May 20) 386 (2-3) 219-24.
Journal code: 0155157. ISSN: 0014-5793.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U25341; GENBANK-U52218; GENBANK-U52219; GENBANK-U52220; GENBANK-U52221
EM 199607
ED Entered STN: 19960805
Last Updated on STN: 20000303
Entered Medline: 19960725
AB We have cloned an **orphan G protein**-coupled **receptor** from a human pituitary cDNA library using a probe generated by PCR. The cDNA, designated H9, encodes a protein of 613 amino acids that is 45% identical at the amino acid level to the recently cloned human Mel(1a) and Mel(1b) melatonin **receptors**. Structural analyses of the encoded protein and its gene, along with phylogenetic analysis, further show that H9 is closely related to the G **protein**-coupled melatonin **receptor** family. Unusual features of the protein encoded by H9 include a lack of N-linked glycosylation sites and a carboxyl tail >300 amino acids long. H9 transiently expressed in COS-1 cells did not **bind** [125I]melatonin or [3H]melatonin. H9 mRNA is expressed in hypothalamus and pituitary, suggesting that the encoded **receptor** and its natural **ligand** are involved in neuroendocrine function.

L4 ANSWER 19 OF 33 MEDLINE on STN
AN 96190677 MEDLINE Full-text
DN PubMed ID: 8619790
TI Identification of 6H1 as a P2Y purinoceptor: P2Y5.
AU Webb T E; Kaplan M G; Barnard E A
CS Molecular Neurobiology Unit, Royal Free Hospital School of Medicine, London, United Kingdom.
SO Biochemical and biophysical research communications, (1996 Feb 6) 219 (1) 105-10.
Journal code: 0372516. ISSN: 0006-291X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-L06109
EM 199606
ED Entered STN: 19960620
Last Updated on STN: 19980206
Entered Medline: 19960613
AB We have determined the identity of the **orphan** G- **protein** coupled **receptor** cDNA, 6H1, present in activated chicken T cells, as a subtype of P2Y purinoceptor. This identification is based on first on the degree of sequence identity shared with recently cloned members of the P2Y **receptor** family and second on the pharmacological profile. Upon transient expression in COS-7 cells the 6H1 **receptor** bound the radiolabel [35S]dATP alpha S specifically and with high affinity (Kd, 10 nM). This specific **binding** could be competitively displaced by a range of **ligands** active at P2 purinoceptors, with ATP being the most active (K (i)), 116 nM). Such competition studies have established the following rank order of activity: ATP ADP 2-methylthioATP alpha, beta-methylene ATP, UTP, thus confirming 6H1 as a member of the growing family of P2Y purinoceptors. As the fifth **receptor** of this type to be identified we suggest that it be named P2Y5.

L4 ANSWER 20 OF 33 MEDLINE on STN
AN 96187229 MEDLINE Full-text
DN PubMed ID: 8777583
TI Leukotriene receptors.
AU Metters K M
CS Department of Biochemistry and Molecular Biology, Merck Frosst Centre for Therapeutic Research, Dorval, Quebec, Canada.
SO Journal of lipid mediators and cell signalling, (1995 Oct) 12 (2-3) 413-27. Ref: 126
Journal code: 9430888. ISSN: 0929-7855.
CY Netherlands
DT Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LA English
FS Priority Journals
EM 199609
ED Entered STN: 19960924
Last Updated on STN: 19970203
Entered Medline: 19960919
AB The current challenge in research on leukotriene **receptors** is to clone these molecules. Traditional protein purification approaches have not been successful in providing sequence information. Solubilization of cys-LT1 has been achieved but results in the dissociation of G- **proteins** and the loss of high affinity **binding** (Mong et al., 1986b; Mong and Sarau, 1990), while cys-LT2 activity cannot be monitored by other than functional **assays** and there have not been any purification attempts. Partial purification of B-LT has been reported but has not been continued to homogeneity (Sherman et al., 1992; Votta et al., 1990; Miki et al., 1990). Nor have attempts to clone these **receptors** through either homology **screening** or expression cloning been successful. The cloning of the prostanoid **receptors**, described in detail elsewhere in this volume, has shown that these **receptors** belong to a distinct family within the **G-protein**-coupled **receptor** superfamily. It is probable, therefore, that the leukotriene **receptors** will also belong to a separate group within this superfamily since phylogenic comparisons have shown that **receptors** displaying high affinity for structurally related **ligands** exist as discrete families. Recently, a human cDNA encoding an **orphan** FMLP-related **receptor** cloned from HL60 cells of myeloid lineage was identified as the **receptor** for another eicosanoid, lipoxin A (Fiore et al., 1994). FMLP has a similar profile of biological actions to LTB4. Moreover, LTD4 showed a high degree of cross-reactivity with this **receptor** with an affinity only 20-fold less that of lipoxin A, although LTB4 was inactive. It remains to be determined whether the leukotriene **receptors** will fall into this class of **receptors.** The cloning of the leukotriene **receptors** will allow identification of the different **receptor** types and subtypes and potentially splice variants. Evaluation of currently developed **antagonists** at these **receptor** types could also open the way for novel therapies for inflammatory conditions.

L4 ANSWER 21 OF 33 MEDLINE on STN
AN 96055113 MEDLINE Full-text
DN PubMed ID: 7481766
TI Orphanin FQ: a neuropeptide that activates an opioidlike G **protein**-coupled receptor.
AU Reinscheid R K; Nothacker H P; Bourson A; Ardati A; Henningsen R A; Bunzow J R; Grandy D K; Langen H; Monsma F J Jr; Civelli O
CS Pharma Division, Hoffmann-La Roche AG, Basel, Switzerland.
NC DA 08562 (NIDA)
DA 09620 (NIDA)
SO Science, (1995 Nov 3) 270 (5237) 792-4.

Journal code: 0404511. ISSN: 0036-8075.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199512
ED Entered STN: 19960124
Last Updated on STN: 20000303
Entered Medline: 19951206
AB A heptadecapeptide was identified and purified from porcine brain tissue as a **ligand** for an **orphan** heterotrimeric GTP- **binding** protein (**G protein**)-coupled **receptor** (LC132) that is similar in sequence to opioid **receptors.** This peptide, orphanin FQ, has a primary structure reminiscent of that of opioid peptides. Nanomolar concentrations of orphanin FQ inhibited forskolin-stimulated adenylyl cyclase activity in cells transfected with LC132. This inhibitory activity was not affected by the addition of opioid **ligands**, nor did the peptide activate opioid **receptors.** Orphanin FQ bound to its **receptor** in a saturable manner and with high affinity. When injected intracerebroventricularly into mice, orphanin FQ caused a decrease in locomotor activity but did not induce analgesia in the hot-plate test. However, the peptide produced hyperalgesia in the tail-flick **assay** . Thus, orphanin FQ may act as a transmitter in the brain by modulating nociceptive and locomotor behavior.

L4 ANSWER 22 OF 33 MEDLINE on STN
AN 96032699 MEDLINE Full-text
DN PubMed ID: 7559404
TI Identification of dynorphins as endogenous ligands for an opioid receptor-like **orphan** receptor.
AU Zhang S; Yu L
CS Department of Medical and Molecular Genetics, Indiana University School of Medicine, Indianapolis 46202, USA.
NC DA09116 (NIDA)
DA09444 (NIDA)
NS28190 (NINDS)
+
SO Journal of biological chemistry, (**1995 Sep 29**) 270 (39) 22772-6.
Journal code: 2985121R. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199511
ED Entered STN: 19951227
Last Updated on STN: 19970203
Entered Medline: 19951106
AB To identify the endogenous **ligands** for a cloned **orphan receptor** that shares high degrees of sequence homology with opioid **receptors**, this **orphan receptor** was expressed in Xenopus oocytes and in mammalian cell lines CHO-K1 and HEK-293. The coupling of the **receptor** to a **G protein** -activated K+ channel was used as a functional **assay** in oocytes. Endogenous opioid peptide dynorphins were found to activate the K+ channel by stimulating the **orphan receptor.** This activation was dose-dependent, with EC50 values at 45 and 37 nM for dynorphin A and dynorphin A-(1-13), respectively. The dynorphin effect was antagonized by the non-selective opioid **antagonist** naloxone but at rather high concentrations in the micromolar range. Naloxone also caused a rightward shift of the dose-response curve for dynorphin A, suggesting a competitive antagonism mechanism. In transiently transfected cells, 5 microM dynorphin A-(1-13) inhibited the forskolin-stimulated cyclic AMP increase by 51 and 35% in

CHO-K1 and HEK-293 cells, respectively. Other classes of endogenous opioids, i.e. enkephalins and endorphins, caused very little activation of this **receptor**. These results suggest that this **orphan receptor** is a member of the opioid **receptor** family and that dynorphins are endogenous **ligands** for this **receptor**.

L4 ANSWER 23 OF 33 MEDLINE on STN
AN 95374679 MEDLINE Full-text
DN PubMed ID: 7646814
TI Cloning, chromosomal localization, and RNA expression of a human beta chemokine receptor-like gene.
AU Combadiere C; Ahuja S K; Murphy P M
CS Laboratory of Host Defenses, National Institute of Allergy and Infectious Diseases, Bethesda, MD 20892, USA.
SO DNA and cell biology, (**1995 Aug**) 14 (8) 673-80.
Journal code: 9004522. ISSN: 1044-5498.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-U28934
EM 199509
ED Entered STN: 19951005
Last Updated on STN: 19951005
Entered Medline: 19950925
AB A human cDNA encoding a putative G **protein**-coupled **receptor** designated chemokine beta **receptor**-like 1 (CMKBRL1) was isolated from an eosinophilic leukemia library. Its deduced sequence is approximately 40% identical to previously cloned **receptors** for the beta chemokines macrophage inflammatory protein-1 alpha (MIP-1 alpha), RANTES, and monocyte chemoattractant protein-1 (MCP-1), which are chemoattractants for blood leukocytes, and is 83% identical to the product of the **orphan** rat cDNA RBS 11. Like the MIP-1 alpha/RANTES **receptor**, CMK-BRL1 is encoded by a small, single-copy gene that maps to chromosome 3p21 and is expressed in leukocytes. However, two **screening assays** with a broad panel of chemokines failed to identify its **ligand**. CMKBRL1 mRNA was detectable by Northern blot hybridization in neutrophils and monocytes, but not eosinophils, and was also found in eight solid organs that were tested with particularly high expression in brain. The RNA distribution of the known beta chemokine **receptors** was overlapping but distinct from that of CMKBRL1. MIP-1 alpha/RANTES **receptor** mRNA was detectable in neutrophils, monocytes, eosinophils, and in all eight solid organs tested, with particularly high expression in placenta, lung, and liver. MCP-1 **receptor** mRNA was found in monocytes, lung, liver, and pancreas. These results suggest that the **ligand** for the putative CMKBRL1 **receptor** is a beta chemokine that targets both neutrophils and monocytes. Moreover, the RNA distributions suggest that CMKBRL1, the MIP-1 alpha/RANTES **receptor**, and the MCP-1 **receptor** may have both overlapping and distinct biological roles.

L4 ANSWER 24 OF 33 MEDLINE on STN
AN 95366960 MEDLINE Full-text
DN PubMed ID: 7639700
TI Molecular cloning of an **orphan G-protein** -coupled receptor that constitutively activates adenylate cyclase.
AU Eggerickx D; Denef J F; Labbe O; Hayashi Y; Refetoff S; Vassart G; Parmentier M; Libert F
CS Institut de Recherche Interdisciplinaire, Faculte de Medecine, Universite Libre de Bruxelles, Belgium.
NC DK 15070 (NIDDK)

SO Biochemical journal, (1995 Aug 1) 309 (Pt 3) 837-43.
Journal code: 2984726R. ISSN: 0264-6021.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
OS GENBANK-X83956
EM 199509
ED Entered STN: 19950921
Last Updated on STN: 20020613
Entered Medline: 19950913
AB A human gene encoding an **orphan G-protein** -coupled **receptor** named ACCA (adenylate cyclase constitutive activator) was isolated from a genomic library using as a probe a DNA fragment obtained by low-stringency PCR. Human ACCA (hACCA) is a protein of 330 amino acids that exhibits all the structural hallmarks of the main family of **G-protein**-coupled **receptors**. Expression of hACCA resulted in a dramatic stimulation of adenylate cyclase, similar in amplitude to that obtained with other Gs-coupled **receptors** fully activated by their respective **ligands**. This stimulation was obtained in a large variety of stable cell lines derived from various organs, and originating from different mammalian species. hACCA was found to be the human homologue of a recently reported mouse **orphan receptor** (GPCR21). The mouse ACCA (mACCA) was therefore recloned by PCR, and expression of mACCA in Cos-7 cells demonstrated that the mouse **receptor** behaved similarly as a constitutive activator of adenylate cyclase. It is not known presently whether the stimulation of adenylate cyclase is the result of a true constitutive activity of the **receptor** or, alternatively, is the consequence of a permanent stimulation by a ubiquitous **ligand**. The tissue distribution of mACCA was determined by RNase protection **assay**. Abundant transcripts were found in the brain, whereas lower amounts were detected in testis, ovary and eye. Various hypotheses concerning the constitutive activity of ACCA and their potential biological significance are discussed.

L4 ANSWER 25 OF 33 MEDLINE on STN
AN 95171461 MEDLINE Full-text
DN PubMed ID: 7867073
TI The anticoagulation factor protein S and its relative, Gas6, are ligands for the Tyro 3/Axl family of receptor tyrosine kinases.
AU Stitt T N; Conn G; Gore M; Lai C; Bruno J; Radziejewski C; Mattsson K; Fisher J; Gies D R; Jones P F; +
CS Regeneron Pharmaceuticals, Incorporated, Tarrytown, New York 10591.
SO Cell, (1995 Feb 24) 80 (4) 661-70.
Journal code: 0413066. ISSN: 0092-8674.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199503
ED Entered STN: 19950407
Last Updated on STN: 20000303
Entered Medline: 19950328
AB We report the identification of **ligands** for Tyro 3 (alternatively called Sky, rse, brt, or tif) and Axl (alternatively, Ark or UFO), members of a previously **orphan** family of **receptor**-like tyrosine kinases. These **ligands** correspond to protein S, a protease regulator that is a potent anticoagulant, and Gas6, a protein related to protein S but lacking any known function. Our results are reminiscent of recent findings that the procoagulant thrombin, a protease that drives clot formation by cleaving fibrinogen to form fibrin, also **binds** and activates intracellular signaling via a G **protein**-coupled cell surface

receptor. Proteases and protease regulators that also activate specific cell surface receptors may serve to integrate coagulation with associated cellular responses required for tissue repair and growth, as well as to coordinate protease cascades and associated cellular responses in other systems, such as those involved in growth and remodeling of the nervous system.

L4 ANSWER 26 OF 33 MEDLINE on STN
AN 91266895 MEDLINE Full-text
DN PubMed ID: 1646713
TI The orphan receptor cDNA RDC7 encodes an A1 adenosine receptor.
AU Libert F; Schiffmann S N; Lefort A; Parmentier M; Gerard C; Dumont J E; Vanderhaeghen J J; Vassart G
CS Institut de Recherche Interdisciplinaire, Faculte de Medecine, Universite Libre de Bruxelles, Belgium.
SO EMBO journal, (1991 Jul) 10 (7) 1677-82.
Journal code: 8208664. ISSN: 0261-4189.
CY ENGLAND: United Kingdom
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals
EM 199107
ED Entered STN: 19910811
Last Updated on STN: 20000303
Entered Medline: 19910724
AB The extensive amino acid sequence conservation among G protein-coupled receptors has been exploited to clone new members of this large family by homology screening or by PCR. Out of four such receptor cDNAs we cloned recently, RDC7 corresponds to a relatively abundant transcript in the brain cortex, the thyroid follicular cell and the testis. We have now identified RDC7 as an A1 adenosine receptor. The A1 agonist CPA [N6-cyclopentyladenosine] decreased by 80% cAMP accumulation in forskolin-stimulated CHO cells stably transfected with RDC7. Specific binding of another A1 adenosine agonist, [3H]CHA [N6-cyclohexyladenosine], was demonstrated on membranes from Cos cells transfected with a pSVL construct harbouring the RDC7 cDNA insert. The binding characteristics were similar to those of the natural brain A1 receptor. The recombinant and the natural receptors behaved also in the same way in displacement experiments involving a series of A1 adenosine agonists. The binding characteristics of RDC7 were compared to those of RDC8, another orphan receptor recently identified as an A2 adenosine receptor. The two molecular species RDC7 and RDC8 correspond clearly to the A1 and A2 receptor entities defined hitherto on a purely pharmacological basis.

L4 ANSWER 27 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 1998:299739 SCISEARCH Full-text
GA The Genuine Article (R) Number: ZG655
TI New aspects of G-protein-coupled receptor signalling and regulation
AU Milligan G (Reprint)
CS UNIV GLASGOW, INST BIOMED & LIFE SCI, DIV BIOCHEM & MOL BIOL, MOL PHARMACOL GRP, GLASGOW G12 8QQ, LANARK, SCOTLAND (Reprint)
CYA SCOTLAND
SO TRENDS IN ENDOCRINOLOGY AND METABOLISM, (JAN-FEB 1998) Vol. 9, No. 1, pp. 13-19.
Publisher: ELSEVIER SCIENCE INC, 655 AVENUE OF THE AMERICAS, NEW YORK, NY 10010.
ISSN: 1043-2760.

DT General Review; Journal
FS LIFE
LA English
REC Reference Count: 52
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB Research on the structure, regulation and signalling properties of the family of seven-transmembrane-helix, heterotrimeric guanine nucleotide-**binding** protein (**G-protein**)-coupled **receptors** (**GPCRs**) continues at a frantic pace. This reflects their central role in transmission of hormone- and neurotransmitter-encoded information across the plasma membrane of cells. The location of the **ligand-binding** sites on the extracellular face of the membrane has made them obvious targets for therapeutic intervention in a wide range of conditions resulting from endocrine imbalance. Furthermore, based on the identification of many novel **GPCR** sequences emerging from expressed sequence tags (ESTs) and other DNA sequencing programmes, it has become clear that the **GPCR** family is likely to be considerably larger than appreciated in even the recent past. Although neither the natural **ligands** nor synthetic pharmaceuticals have yet been identified for these so-called '**orphan**' **GPCRs**, they offer the potential for a plethora of new therapeutic targets. Within a short review, it is impossible to cover all the current developments in this field and the topics selected represent a personal view of recent high-lights of areas that provide both novel and general insights into the function and regulation of **GPCRs**.

L4 ANSWER 28 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 1998:240418 SCISEARCH Full-text
GA The Genuine Article (R) Number: ZC567
TI A human gene encodes a putative G **protein**-coupled receptor highly expressed in the central nervous system
AU Donohue P J; Shapira H; Mantey S A; Hampton L L; Jensen R T; Battey J F (Reprint)
CS NATL INST DEAFNESS & OTHER COMMUN DISORDERS, MOL BIOL LAB, NIH, ROCKVILLE, MD 20850 (Reprint); NATL INST DEAFNESS & OTHER COMMUN DISORDERS, MOL BIOL LAB, NIH, ROCKVILLE, MD 20850; TEL AVIV UNIV, SACKLER FAC MED, DEPT PHYSIOL & PHARMACOL, IL-69978 RAMAT AVIV, ISRAEL; NIDDKD, DIGEST DIS BRANCH, NIH, BETHESDA, MD 20892
CYA USA; ISRAEL
SO MOLECULAR BRAIN RESEARCH, (**FEB 1998**) Vol. 54, No. 1, pp. 152-160.
Publisher: ELSEVIER SCIENCE BV, PO BOX 211, 1000 AE AMSTERDAM, NETHERLANDS.
ISSN: 0169-328X.
DT Article; Journal
FS LIFE
LA English
REC Reference Count: 16
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB The mammalian bombesin (Bn)-like neuropeptide **receptors** gastrin-releasing peptide **receptor** (GRP-R) and neuromedin B **receptor** (NMB-R) transduce a variety of physiological signals that regulate secretion, growth, muscle contraction, chemotaxis and neuromodulation. We have used reverse transcription-polymerase chain reaction (PCR) to isolate a cDNA from human brain mRNA, **GPCR** /CNS, that encodes a putative G **protein**-coupled **receptor** (**GPCR**) based upon the presence of the paradigmatic seven heptahelical transmembrane domains in its predicted amino acid sequence. Analysis of the deduced protein sequence of **GPCR**/CNS reveals this putative **receptor** to be 98% identical to the deduced amino acid sequence of a recently reported

gene product and minimally identical (similar to 23%) to both murine GRP-R and human endothelin-B (ET-B) **receptor**. Our deduced protein sequence differs at 12 positions, scattered throughout the open reading frame, relative to the original sequence. A 3.7 kb **GPCR**/CNS mRNA species is expressed in vivo in a tissue-specific manner, with highest levels detected in brain and spinal cord, lower levels found in testis, placenta and liver, but no detectable expression observed in any other tissue. Analysis of **GPCR**/CNS genomic clones reveals that the human gene contains one intron that is about 21 kb in length that divides the coding region into two exons and maps to human chromosome 7q31. No specific **binding** is observed with either a newly identified **ligand** (DTyr(6),beta Ala(11),Phe(13),Nle(14)]Bn-(6-14)) having high affinity for all Bn **receptor** subtypes or Bn after **GPCR**/CNS is stably expressed in fibroblasts. No elevation in inositol trisphosphate is observed after the application of micromolar levels of either DPhe(6),beta Ala(11),Phe(13),Nle(14)]Bn-(6-14) or Bn, a concentration of **agonist** known to activate all four known Bn **receptor** subtypes. When **GPCR**/CNS is expressed in Xenopus oocytes, no activation of the calcium-dependent chloride channel is detected despite the addition of micromolar levels of Bn peptide **agonists**. We conclude that the natural **ligand** for this **receptor** is none of the known naturally occurring Bn-like peptides and the true **agonist** for **GPCR**/CNS remains to be elucidated. (C) 1998 Elsevier Science B.V.

L4 ANSWER 29 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 95:810225 SCISEARCH Full-text
GA The Genuine Article (R) Number: TF351
TI CLONING, SEQUENCING AND TISSUE DISTRIBUTION OF 2 RELATED G-**PROTEIN**-COUPLED RECEPTOR CANDIDATES EXPRESSED PROMINENTLY IN HUMAN LUNG-TISSUE
AU AN S Z (Reprint); TSAI C; GOETZL E J
CS UNIV CALIF SAN FRANCISCO, DEPT MED, DIV ALLERGY & IMMUNOL, SAN FRANCISCO, CA, 94143 (Reprint); UNIV CALIF SAN FRANCISCO, DEPT IMMUNOL MICROBIOL, SAN FRANCISCO, CA, 94143
CYA USA
SO FEBS LETTERS, (13 **NOV** 1995) Vol. 375, No. 1-2, pp. 121-124. ISSN: 0014-5793.
DT Article; Journal
FS LIFE
LA ENGLISH
REC Reference Count: 22
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB A novel G **protein**-coupled **receptor**, named GPR12A, was cloned by a PCR strategy using degenerate primers designed from sequences conserved among **receptors** for inflammatory mediators, **Screening** of a human lung cDNA library with GPR12A as a probe also identified a closely-related cDNA (GPR6C.1) that has been previously reported as GPR4 [13], GPR12A and GPR6C.1 are 46.1% identical in amino acid sequence, but are less than 33% identical to any other known **receptors**, Northern analysis revealed that they are expressed prominently in the lung, Although the **ligands** for GPR12A and GPR6C.1 are unknown, their similarity suggests that they are **receptors** for **ligands** of similar or identical chemical nature.

L4 ANSWER 30 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 95:161028 SCISEARCH Full-text
GA The Genuine Article (R) Number: QH537
TI **G-PROTEIN**-COUPLED **ORPHAN** RECEPTORS

AU PARMENTIER M (Reprint); LIBERT F; VASSART G
CS FREE UNIV BRUSSELS, IRIBHN, FONDS NATL RECH SCI, CAMPUS ERASME, 808 ROUTE LENNIK, B-1070 BRUSSELS, BELGIUM (Reprint)
CYA BELGIUM
SO M S-MEDECINE SCIENCES, (**FEB 1995**) Vol. 11, No. 2, pp. 222-231.
ISSN: 0767-0974.
DT General Review; Journal
FS LIFE
LA French
REC No References Keyed
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS
AB **G protein**-coupled **receptors** are encoded by one of the mammalian largest gene families. These **receptors** share a common transmembrane organization, respond to a large variety of structurally: different **ligands**, and regulate intracellular enzymes and channels through heterotrimeric **G proteins** . The first genes encoding **receptors** of this family were isolated following the purification of the protein and sequencing of peptides generated by proteolytic cleavage. Subsequently, a number of **G protein**-coupled **receptor** genes were cloned through expression **screening** procedures based either on the **binding** of a specific **ligand**, or on the functional coupling of the recombinant **receptor** to a transduction cascade. Most members in this gene family were however obtained by homology cloning, using cross-hybridization or low-stringency polymerase chain reaction. The result is the availability of more than 140 **receptor** types and subtypes (olfactory **receptors** excluded). With a few exceptions, all pharmacologically well-defined **receptors** have now been cloned. Molecular cloning confirmed the existence of poorly characterized subtypes and uncovered other unsuspected subtypes. Genes encoding uncharacterized **receptors** have also been made available, either related to identified subfamilies, or defining new subfamilies by themselves. These so-called << **orphan** >> **receptors** are waiting for the identification of the corresponding **ligands** and of their biological function. The availability of cloned human **receptors** is expected to speed up the search for more specific drugs. Determination of the three dimensional structure of selected recombinant **receptors**, and computer modelling of **receptor-ligand** inter action and **ligand** -mediated **receptor** activation will probably allow in the long run the rational design of specific **agonists** and **antagonists.** The increasing number of reported **orphan receptors** will certainly be instrumental in the discovery of new regulatory pathways linking cells, with the potential of opening new avenues in pharmacology.

L4 ANSWER 31 OF 33 SCISEARCH COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 94:196245 SCISEARCH Full-text
GA The Genuine Article (R) Number: NC117
TI CDNA CLONING OF A G-**PROTEIN**-COUPLED RECEPTOR EXPRESSED IN RAT SPINAL-CORD AND BRAIN RELATED TO CHEMOKINE RECEPTORS
AU HARRISON J K (Reprint); BARBER C M; LYNCH K R
CS UNIV FLORIDA, COLL MED, DEPT PHARMACOL & THERAPEUT, BOX 100267, GAINESVILLE, FL, 32610 (Reprint); UNIV VIRGINIA, SCH MED, DEPT PHARMACOL, CHARLOTTESVILLE, VA, 22908; UNIV VIRGINIA, SCH MED, DEPT BIOCHEM, CHARLOTTESVILLE, VA, 22908
CYA USA
SO NEUROSCIENCE LETTERS, (**14 MAR 1994**) Vol. 169, No. 1-2, pp. 85-89.
ISSN: 0304-3940.
DT Article; Journal
FS LIFE
LA ENGLISH

REC Reference Count: 18
ABSTRACT IS AVAILABLE IN THE ALL AND IALL FORMATS

AB A series of cDNAs and a genomic clone (named RBS11) were isolated, from a variety of rat brainstem, pituitary and/or spinal cord cDNA libraries and a genomic library by low-stringency hybridization **screening** with a rat angiotensin **receptor** cDNA. The RBS11 protein, as conceptualized from these DNAs, is a novel member of the rhodopsin family of the **G-protein**-coupled **receptor** (GCR) superfamily. Comparison of RBS11 to other members of the GCR superfamily suggests that the RBS11 protein might be a **receptor** for a peptide **ligand** in the chemokine family. The RBS11 protein sequence is unusual in that it is without N-linked glycosylation consensus sequences in the putative exofacial regions. Northern analysis indicates that the mRNA for RBS11 accumulates widely and unevenly in the adult rat, with the mRNA being most prominent in extracts of spinal cord, brain, kidney, gut, uterus and testes.

L4 ANSWER 32 OF 33 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 1997:207816 BIOSIS Full-text
DN PREV199799507019
TI Functional coupling of the nociceptin/orphanin FQ receptor with the **G-protein**-activated K+ (GIRK) channel.
AU Ikeda, Kazutaka [Reprint author]; Kobayashi, Katsunori; Kobayashi, Toru; Ichikawa, Tomio; Kumanishi, Toshiro; Kishida, Haruo; Yano, Ryoji; Manabe, Toshiya
CS Lab. Cell. Inf. Processing, Frontier Res. Program, Inst. Phys. Chem. Res., 2-1 Hirosawa, Wako, Saitama 351-01, Japan
SO Molecular Brain Research, (1997) Vol. 45, No. 1, pp. 117-126.
CODEN: MBREE4. ISSN: 0169-328X.
DT Article
LA English
ED Entered STN: 12 May 1997
Last Updated on STN: 2 Jun 1997
AB Nociceptin/orphanin FQ is a heptadecapeptide which was recently isolated from brains. It induces hyperalgesia, in contrast to the analgesic effects of opioid **ligands**, although it and its **receptor** structurally resemble opioid peptides and opioid **receptors**, respectively. To investigate the molecular mechanism underlying nociceptin/orphanin FQ actions, we performed Xenopus oocyte expression **assays**, in situ hybridization histochemistry and electrophysiological analyses of neurons. We found that the nociceptin/orphanin FQ **receptor** is functionally coupled with the **G-protein**-activated K+ (GIRK) channel in Xenopus oocytes, and that the **receptor** mRNA and GIRK1 mRNA co-exist in various neurons, including hippocampal pyramidal cells. Furthermore, we found that nociceptin/orphanin FQ induces hyperpolarizing currents via inward-rectifier K+ channels in hippocampal pyramidal cells, suggesting that the nociceptin/orphanin FQ **receptor** couples with the GIRK channel in this region. We conclude that the nociceptin/orphanin FQ **receptor** couples with the GIRK channel in various neurons, including hippocampal pyramidal cells, thereby modulating neuronal excitability.

L4 ANSWER 33 OF 33 BIOSIS COPYRIGHT (c) 2005 The Thomson Corporation. on STN
AN 1995:381269 BIOSIS Full-text
DN PREV199598395569
TI Studies of **ligand binding** and signal transduction pathway of the chimeric **receptor** of IL-8 **receptor** and LESTR.
AU Na, S.; Ogborne, K. T.; Hinkel, C. A.; Neote, K. S.; Hanke, J. H.

CS Central Res., Pfizer Inc., Groton, CT, USA
SO 9TH INTERNATIONAL CONGRESS OF IMMUNOLOGY. (1995) pp. 111. The 9th International Congress of Immunology.
Publisher: 9th International Congress of Immunology, San Francisco, California, USA.
Meeting Info.: Meeting Sponsored by the American Association of Immunologists and the International Union of Immunological Societies. San Francisco, California, USA. July 23-29, 1995.
DT Conference; (Meeting)
Conference; Abstract; (Meeting Abstract)
LA English
ED Entered STN: 1 Sep 1995
Last Updated on STN: 1 Sep 1995

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FILE CONTAINS CURRENT INFORMATION.
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(FILE 'HOME' ENTERED AT 11:59:37 ON 15 JAN 2005)

FILE 'MEDLINE, SCISEARCH, EMBASE, BIOSIS' ENTERED AT 11:59:56 ON 15 JAN 2005
L1 2546 S RECEPTOR (P) (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND) (P
L2 912 S L1 AND PY<=1998
L3 284 DUP REM L2 (628 DUPLICATES REMOVED)
L4 33 S L3 AND (GPCR OR G (W) PROTEIN)

FILE 'STNGUIDE' ENTERED AT 12:07:17 ON 15 JAN 2005

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East Search 1/15/2005

Ref #	Hits	Search Query	DBs	Default Operator	Plurals	Time Stamp
L1	157	(hinuma-shuji or hosoya-masaki).in.	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/01/15 16:09
L2	16	l1 and orphan adj receptor	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/01/15 16:12
L3	379	(orphan adj receptor) same (LIGAND OR AGONIST OR ANTAGONIST OR COMPOUND$) same (SCREEN$ OR BIND$ OR ASSAY$)	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/01/15 16:13
L4	42	l3 and @py<="1999"	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/01/15 16:14
L5	58	l2 or l4	US-PGPUB; USPAT; EPO; JPO; DERWENT	OR	OFF	2005/01/15 16:14

East Search **15 January 2005**

US 20040254348 A1	US-PGPUB	20041216	Novel polypeptide and dna thereof
US 20040248790 A1	US-PGPUB	20041209	Novel secretory proteins and dna thereof
US 20040171067 A1	US-PGPUB	20040902	Screening method
US 20040152136 A1	US-PGPUB	20040805	Screening method
US 20040132073 A1	US-PGPUB	20040708	Novel G protein-coupled receptor protein, its DNA and ligand thereof
US 20030092618 A1	US-PGPUB	20030515	Polypeptides, their production and use
US 20030082648 A1	US-PGPUB	20030501	Novel mass receptor-analogous protein and dnas thereof
US 20020168720 A1	US-PGPUB	20021114	G protein coupled receptor proteins, their production and use
US 20020143152 A1	US-PGPUB	20021003	Polypeptides, their production and use
US 20020009771 A1	US-PGPUB	20020124	G PROTEIN COUPLED RECEPTOR PROTEINS, THEIR PRODUCTION AND USE
US 6794491 B1	USPAT	20040921	Polypeptides, their production and use
US 6492324 B1	USPAT	20021210	APJ ligand polypeptides
US 6228984 B1	USPAT	20010508	Polypeptides their production and use
US 6005086 A	USPAT	19991221	Farnesoid activated receptor polypeptides, and nucleic acid encoding the same
US 5989834 A	USPAT	19991123	Uses of nucleic acid encoding neuropeptide Y/peptide YY (Y2) receptors nucleic acid encoding
US 5989537 A	USPAT	19991123	Methods for stimulating granulocyte/macrophage lineage using thrombopoietin
US 5976834 A	USPAT	19991102	cDNA clone HNFJD15 that encodes a novel human 7-transmembrane receptor
US 5958683 A	USPAT	19990928	Screening method using the RZR receptor family
US 5955303 A	USPAT	19990921	Human chemokine receptor-like protein
US 5945279 A	USPAT	19990831	Screening system for identifying compounds that regulate steroid and orphan receptors mediation of DNA transcription
US 5942405 A	USPAT	19990824	Therapeutic and screening methods using C3a receptor and C3a
US 5912335 A	USPAT	19990615	G-protein coupled receptor HUVCT36

US 5871916 A	USPAT	19990216	ECDN protein and DNA encoding the same
US 5866346 A	USPAT	19990202	Methods of using dynorphins as ligands for XOR1 receptor
US 5849485 A	USPAT	19981215	Liver enriched transcription factor
US 5834213 A	USPAT	19981110	Screening system and assay for identifying compounds that regulate steroid and orphan receptors mediation of DNA transcription
US 5824504 A	USPAT	19981020	Human 7-transmembrane receptor and DNA
US 5814479 A	USPAT	19980929	Bsk receptor-like tyrosine kinase
US 5789170 A	USPAT	19980804	Specific co-activator for human androgen receptor
US 5783402 A	USPAT	19980721	Method of identifying ligands and anatgonists of G-protein coupled receptor
US 5747661 A	USPAT	19980505	Retinoid-inducible response elements
US 5714595 A	USPAT	19980203	Mechanism-based screen for retinoid X receptor agonists and antagonists
US 5707800 A	USPAT	19980113	Retinoic acid response elements and assays employing same
US 5705349 A	USPAT	19980106	Methods for preparing polynucleotides encoding orphan receptor ligands
US 5700682 A	USPAT	19971223	Mechanism based screen for retinoid X receptor agonists and antagonists
US 5700650 A	USPAT	19971223	Mechanism-based screen for retinoid X receptor agonists and antagonists
US 5691196 A	USPAT	19971125	Recombinant nucleic acid containing a response element
US 5639616 A	USPAT	19970617	Isolated nucleic acid encoding a ubiquitous nuclear receptor
US 5604115 A	USPAT	19970218	Liver enriched transcription factor
US 5597693 A	USPAT	19970128	Hormone response element compositions and assay
US 5545549 A	USPAT	19960813	DNA encoding a human neuropeptide Y/peptide YY (Y2) receptor and uses thereof
US 5541085 A	USPAT	19960730	Method for preparing orphan receptor ligands
US 5538861 A	USPAT	19960723	DNA encoding a stimulating factor for the axl receptor
US 5532157 A	USPAT	19960702	Host cell line LVIP2.0Zc, useful in assays to identify ligands and ant agonists of G protein-coupled receptors
US 5514578 A	USPAT	19960507	Polynucleotides encoding insect steroid hormone receptor polypeptides and cells transformed with same

JP 2004180668 A	JPO	20040702	METHOD FOR DETERMINING LIGAND
JP 2001128695 A	JPO	20010515	SCREENING METHOD
JP 411315099 A	JPO	19991116	PRODUCTION OF VARIANT TYPE ORPHAN RECEPTOR AND VARIANT TYPE NOCICEPTIN RECEPTOR
JP 10068724 A	JPO	19980310	SCREENING METHOD FOR LC132 (OPIOID) RECEPTOR AGONIST
WO 3071272 A1	EPO	20030828	METHOD OF DETERMINING LIGAND
JP 11315099 A	DERWENT	19991116	A process for preparation of a variant orphan receptor and a variant nociceptin receptor - useful for the development of opioid analgesics with reduced adverse reactions
US 20020143152 A	DERWENT	20021003	New polypeptide ligand for orphan G protein coupled receptors - used for treating disorders of central nervous system, pituitary and pancreas, and for drug screening
US 5597693 A	DERWENT	19970128	Steroid/thyroid hormone receptor DNA-binding domain compsns. - for determining target gene specificity, also methods for identifying ligands that activate orphan receptors and for altering target gene specificity
US 6458926 B	DERWENT	20021001	Heterodimeric complex of retinoid X receptor (RXR) and a silent partner - useful in transduction of novel responses to multiple hormonal signals
US 5541085 A	DERWENT	19960730	Isolation of orphan receptor ligands - by mutagenising transfected cells which express the orphan receptor and obtaining ligands from surviving cells
WO 9313129 A	DERWENT	19930708	Hetero-dimer comprising retinoid X receptor and hormone receptor - enhances or inhibits transcription, used for screening ligands for effect on activity or RXR-contg. hormone receptor hetero-dimers
US 5707800 A	DERWENT	19980113	Retinoic acid response element - used for controlling recombinant DNA expression in cells and for

			screening agonists and antagonists
EP 463081 B for identifying ligands for	DERWENT	19980520	Steroid and thyroid hormone response elements - used to develop assays orphan hormone receptors

Day : Saturday
Date: 1/15/2005

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Inventor Information for 10/069228

Inventor Name	City	State/Country
HINUMA, SHUJI	IBARAKI	JAPAN
HOSOYA, MASAKI	IBARAKI	JAPAN

Appln Info | Contents | Petition Info | Atty/Agent Info | Continuity Data | Foreign Data

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Inventor Name Search Result

Your Search was:

Last Name = HINUMA
First Name = SHUJI

Application#	Patent#	Status	Date Filed	Title	Inventor Name 51
60017954	Not Issued	159	05/20/1996	NOVEL HUMAN G-PROTEIN COUPLED RECEPTOR	HINUMA , SHUJI
60017915	Not Issued	159	05/16/1996	NOVEL HUMAN G-PROTEIN COUPLED RECEPTOR (HIBCD07)	HINUMA , SHUJI
10914735	Not Issued	019	08/09/2004	G-PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES	HINUMA, SHUJI
10719587	Not Issued	020	11/21/2003	NOVEL G PROTEIN-COUPLED RECEPTOR PROTEIN, ITS DNA AND LIGAND THEREOF	HINUMA, SHUJI
10706703	Not Issued	160	11/12/2003	G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES	HINUMA, SHUJI
10505486	Not Issued	019	01/01/0001	METHOD OF DETERMINING LIGAND	HINUMA, SHUJI
10504726	Not Issued	020	08/13/2004	NOVEL SCREENING METHOD	HINUMA, SHUJI
10498848	Not Issued	019	01/01/0001	METHOD OF ANALYZING GENE EXPRESSION	HINUMA, SHUJI
10488523	Not Issued	030	03/02/2004	USE OF G PROTEIN-COUPLED RECEPTOR PROTEIN	HINUMA, SHUJI
10488480	Not Issued	030	06/15/2004	NOVEL SECRETORY PROTEINS AND DNA THEREOF	HINUMA, SHUJI
10488038	Not Issued	030	02/27/2004	SCREENING METHOD	HINUMA, SHUJI
10487634	Not Issued	071	02/24/2004	NOVEL RFRP-3 AND DNA THEREOF	HINUMA, SHUJI
10480739	Not Issued	030	12/12/2003	NOVEL LIGAND AND DNA THEREOF	HINUMA, SHUJI
10460614	Not Issued	160	06/12/2003	G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES	HINUMA, SHUJI

				AND POLYNUCLEOTIDES	
10332738	Not Issued	030	01/10/2003	NOVEL POLYPEPTIDE AND DNA THEREOF	HINUMA, SHUJI
10309870	Not Issued	160	12/04/2002	G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES	HINUMA, SHUJI
10278087	Not Issued	030	10/22/2002	G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION, AND USE THEREOF	HINUMA, SHUJI
10239321	Not Issued	030	09/19/2002	PEPTIDE DERIVATIVES	HINUMA, SHUJI
10221841	Not Issued	041	09/12/2002	NOVEL MASS RECEPTOR-ANALOGOUS PROTEIN AND DNAS THEREOF	HINUMA, SHUJI
10203015	Not Issued	030	09/23/2002	SCREENING METHOD	HINUMA, SHUJI
10185465	Not Issued	160	06/27/2002	G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES	HINUMA, SHUJI
10184722	Not Issued	030	06/27/2002	POLYPEPTIDES, THEIR PRODUCTION AND USE	HINUMA, SHUJI
10184426	Not Issued	041	06/28/2002	G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF	HINUMA, SHUJI
10168048	Not Issued	030	06/12/2002	NOVEL POLYPEPTIDE AND DNA THEREOF	HINUMA, SHUJI
10148497	Not Issued	083	05/28/2002	SCREENING METHOD	HINUMA, SHUJI
10130584	Not Issued	041	05/16/2002	USE OF PEPTIDE	HINUMA, SHUJI
10111896	Not Issued	041	04/30/2002	GHSR LIGAND POLYPEPTIDES AND DNAS THEREOF	HINUMA, SHUJI
10090569	Not Issued	041	03/04/2002	GALANIN RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF	HINUMA, SHUJI
10069228	Not Issued	071	02/21/2002	SCREENING METHOD	HINUMA, SHUJI
10067477	Not Issued	161	02/04/2002	G PROTEIN COUPLED RECEPTOR PROTEINS, THEIR PRODUCTION AND USE	HINUMA, SHUJI
10044592	Not Issued	161	01/10/2002	POLYPEPTIDES, THEIR PRODUCTION AND USE	HINUMA, SHUJI

09942367	Not Issued	160	08/29/2001	G PROTEIN-COUPLED RECEPTOR POLYPEPTIDES AND POLYNUCLEOTIDES	HINUMA, SHUJI
09929752	Not Issued	161	08/14/2001	G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF	HINUMA, SHUJI
09868885	Not Issued	041	06/22/2001	USE OF PEPTIDE	HINUMA, SHUJI
09787658	Not Issued	161	03/20/2001	APJ LIGAND PEPTIDE DERIVATIVES	HINUMA, SHUJI
09744226	Not Issued	161	01/22/2001	NOVEL G PROTEIN COUPLED RECEPTOR AND ITS DNA	HINUMA, SHUJI
09716147	Not Issued	092	11/17/2000	POLYPEPTIDES, THEIR PRODUCTION AND USE	HINUMA, SHUJI
09700643	Not Issued	094	02/02/2001	ANTIBODY AND USE THEREOF	HINUMA, SHUJI
09576290	6794491	150	05/23/2000	POLYPEPTIDES, THEIR PRODUCTION AND USE	HINUMA, SHUJI
09505396	Not Issued	161	02/16/2000	RECEPTOR EXPRESSION CELLS AND USE THEREOF	HINUMA, SHUJI
09481535	6180792	150	01/12/2000	CONDENSED-RING THIOPHENE DERIVATIVES AND THIENOPYRIMIDE DERIVATIVES, THEIR PRODUCTION AND USE	HINUMA, SHUJI
09461436	6538107	150	12/14/1999	G PROTEIN COUPLED RECEPTOR PROTEIN PRODUCTION, AND USE THEREOF	HINUMA, SHUJI
09446543	Not Issued	161	12/20/1999	PROLACTIN SECRETION MODULATOR	HINUMA, SHUJI
09403639	Not Issued	161	10/25/1999	POLYPEPTIDES, THEIR PRODUCTION AND USE	HINUMA , SHUJI
09390799	Not Issued	161	09/07/1999	NOVEL-HUMAN G-PROTEIN COUPLED RECEPTOR PROTEIN AND ITS DNA	HINUMA , SHUJI
08423691	5677184	150	04/18/1995	CHO CELLS THAT EXPRESS HUMAN LH-RH RECEPTOR	HINUMA , SHUJI
08386354	5556946	150	02/08/1995	INTERLEUKIN-2/VIRAL ANTIGEN PROTEIN CHIMERS	HINUMA , SHUJI
08288663	5879896	150	08/09/1994	A METHOD OF SCREENING FOR INHIBITORS OF HUMAN THYROTROPIN RELEASING HO RMUNE (TRH) RECEPTOR	HINUMA , SHUJI

08181968	Not Issued	166	01/18/1994	HUMAN INTERLEUKIN 2	HINUMA , SHUJI
08086429	Not Issued	164	06/30/1993	ANTIGEN/INTERLEUKIN-2 FUSION PROTEINS	HINUMA , SHUJI
07181362	Not Issued	166	04/13/1988	ASSAY FOR ANTI-PRE-S ANTIBODY	HINUMA , SHUJI

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Last Name = HOSOYA
First Name = MASAKI

Application#	Patent#	Status	Date Filed	Title	Inventor Name 36
10719587	Not Issued	020	11/21/2003	NOVEL G PROTEIN-COUPLED RECEPTOR PROTEIN, ITS DNA AND LIGAND THEREOF	HOSOYA, MASAKI
10504726	Not Issued	020	08/13/2004	NOVEL SCREENING METHOD	HOSOYA, MASAKI
10488523	Not Issued	030	03/02/2004	USE OF G PROTEIN-COUPLED RECEPTOR PROTEIN	HOSOYA, MASAKI
10488480	Not Issued	030	06/15/2004	NOVEL SECRETORY PROTEINS AND DNA THEREOF	HOSOYA, MASAKI
10488038	Not Issued	030	02/27/2004	SCREENING METHOD	HOSOYA, MASAKI
10487634	Not Issued	071	02/24/2004	NOVEL RFRP-3 AND DNA THEREOF	HOSOYA, MASAKI
10474481	Not Issued	030	10/08/2003	SCREENING METHOD	HOSOYA, MASAKI
10239321	Not Issued	030	09/19/2002	PEPTIDE DERIVATIVES	HOSOYA, MASAKI
10203015	Not Issued	030	09/23/2002	SCREENING METHOD	HOSOYA, MASAKI
10184722	Not Issued	030	06/27/2002	POLYPEPTIDES, THEIR PRODUCTION AND USE	HOSOYA, MASAKI
10168048	Not Issued	030	06/12/2002	NOVEL POLYPEPTIDE AND DNA THEREOF	HOSOYA, MASAKI
10148497	Not Issued	083	05/28/2002	SCREENING METHOD	HOSOYA, MASAKI
10090569	Not Issued	041	03/04/2002	GALANIN RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF	HOSOYA, MASAKI
10069228	Not Issued	071	02/21/2002	SCREENING METHOD	HOSOYA, MASAKI
10067477	Not	161	02/04/2002	G PROTEIN COUPLED	HOSOYA,

	Issued			RECEPTOR PROTEINS, THEIR PRODUCTION AND USE	MASAKI
09935371	Not Issued	161	08/22/2001	PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF	HOSOYA, MASAKI
09831758	Not Issued	161	05/11/2001	NOVEL G PROTEIN COUPLED RECEPTOR PROTEIN ITS DNA AND LIGAND THEREOF	HOSOYA, MASAKI
09716147	Not Issued	092	11/17/2000	POLYPEPTIDES, THEIR PRODUCTION AND USE	HOSOYA, MASAKI
09576290	6794491	150	05/23/2000	POLYPEPTIDES, THEIR PRODUCTION AND USE	HOSOYA, MASAKI
09261085	6113960	150	03/02/1999	METHOD FOR COATING FOOD PIECES WITH POWDER MATERIAL	HOSOYA , MASAKI
09255518	6492324	150	02/22/1999	APJ LIGAND POLYPEPTIDES	HOSOYA , MASAKI
09201474	6399316	150	11/30/1998	PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF	HOSOYA , MASAKI
09038572	Not Issued	161	03/11/1998	G PROTEIN COUPLED RECEPTOR PROTEIN, PRODUCTION, AND USE THEREOF	HOSOYA , MASAKI
08978525	Not Issued	161	11/25/1997	DNA PRIMER AND A METHOD FOR SCREENING DNAS	HOSOYA , MASAKI
08932325	6058877	150	09/17/1997	APPARATUS FOR SPRINKLING FOOD PIECES WITH POWDER MATERIAL	HOSOYA , MASAKI
08856154	5973117	150	05/14/1997	PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF	HOSOYA , MASAKI
08855213	5892004	150	05/13/1997	METHOD FOR PREPARING PACAP RECEPTOR PROTEIN	HOSOYA , MASAKI
08811897	5858787	150	03/05/1997	DNA ENCODING PACAP RECEPTOR PROTEIN AND METHOD FOR PREPARING SAID PROTEIN	HOSOYA , MASAKI
08796570	Not Issued	161	02/06/1997	G PROTEIN COUPLED RECEPTOR PROTEINS, THEIR PRODUCTION AND USE	HOSOYA , MASAKI
08776971	6228984	150	02/07/1997	POLYPEPTIDES THEIR	HOSOYA ,

				PRODUCTION AND USE	MASAKI
08566037	5756295	150	12/01/1995	DNA PRIMER AND A METHOD FOR SCREENING DNAS	HOSOYA , MASAKI
08550120	5985554	150	10/30/1995	METHOD OF PROVING THE FUNCTION OF PROTEINS OR PEPTIDES ENCODED BY PARTIALLY SEQUENCED CDNAS BY INHIBITING PROTEIN SYNTHESIS WITH ANTISENSE OLIGONUCLEOTIDES	HOSOYA , MASAKI
08540650	6399325	150	10/11/1995	GALANIN RECEPTOR PROTEIN, PRODUCTION AND USE THEREOF	HOSOYA , MASAKI
08513974	6114139	150	09/14/1995	A G- PROTEIN COUPLED RECEPTOR PROTEIN AND A DNA ENCODING THE RECEPTOR	HOSOYA , MASAKI
08288663	5879896	150	08/09/1994	A METHOD OF SCREENING FOR INHIBITORS OF HUMAN THYROTROPIN RELEASING HO RMUNE (TRH) RECEPTOR	HOSOYA , MASAKI
08202986	Not Issued	166	02/25/1994	PACAP RECEPTOR PROTEIN, METHOD FOR PREPARING SAID PROTEIN, AND USE THEREOF	HOSOYA , MASAKI

Inventor Search Completed: No Records to Display.

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